CLAIMS

1. A method of inhibiting signaling between a T cell and a second cell participating in an immune response in a mammal, comprising:
(a) identifying a mammal selected from the group consisting of a mammal with an immune disease or disorder and a mammal in preparation for a tissue graft; and
(b) administering to the mammal an effective amount of a KIM-1 antagonist selected from the group consisting of: (i) a polypeptide comprising a KIM-1 Ig domain, and lacking a transmembrane domain and a KIM-1 cytoplasmic domain; (ii) an anti-KIM-1 antibody; and (iii) an antigen-binding fragment of an anti-KIM-1 antibody.

2. The method of claim 1, wherein the second cell is an antigen presenting cell (APC).

3. The method of claim 1, wherein the T cell is an activated T cell.

4. The method of claim 1, wherein the T cell is a T helper cell.

5. The method of claim 4, wherein the T helper cell is a Th2 cell.

6. The method of claim 1, wherein the T cell is a grafted, donor T cell.

7. The method of claim 1, wherein the APC is selected from the group consisting of a monocyte, a macrophage, a dendritic cell, and a B cell.

8. The method of claim 1, wherein the APC is presenting an autoantigen.

9. The method of claim 1, wherein the polypeptide further comprises a KIM-1 mucin domain.

10. The method of claim 1 wherein the polypeptide further comprises a heterologous moiety.

11. The method of claim 10, wherein the heterologous moiety is selected from the group consisting of an immunoglobulin (Ig) moiety, a serum albumin moiety, a targeting moiety, a reporter moiety, and a purification-facilitating moiety.

12. The method of claim 11, wherein the heterologous moiety is an Ig moiety.

13. The method of claim 12, wherein the Ig moiety is an Fc moiety.

14. The method of claim 1, wherein the polypeptide is conjugated to a polymer.

15. The method of claim 14, wherein the polymer is selected from the group consisting of a polyalkylene glycol, a sugar polymer, and a polypeptide.

16. The method of claim 15, wherein the polymer is a polyalkylene glycol.

17. The method of claim 16, wherein the polyalkylene glycol is polyethylene glycol (PEG).

18. The method of claim 17, wherein the average molecular weight of the polymer is from 2,000 Da to 30,000 Da.

19. The method of claim 18, wherein the average molecular weight of the polymer is from 5,000 Da to 20,000 Da.

20. The method of claim 19, wherein the average molecular weight of the polymer is about 10,000 Da.

21. A method of inhibiting activation of a B cell in a mammal, comprising contacting the B cell with an effective amount of a KIM-1 antagonist selected from the group consisting of: (a) a polypeptide comprising a KIM-1 Ig domain, and lacking a transmembrane domain and a KIM-1 cytoplasmic domain; (b) an anti-KIM-1 antibody; and (c) an antigen-binding fragment of an anti-KIM-1 antibody.

22. The method of claim 21, wherein the activation of the B cell is mediated by an activated T cell.

23. The method of claim 22, wherein the activated T cell is a Th2 cell.

24. The method of claim 22, wherein the activated T cell is a grafted, donor T cell.

25. A method of inhibiting production in a mammal of a subset of antibodies against one or more antigens, comprising administering an effective amount of a KIM-1 antagonist selected from the group consisting of: (a) a polypeptide comprising a KIM-1 Ig domain, and lacking a transmembrane domain and a KIM-1 cytoplasmic domain; (b) an anti-KIM-1 antibody; and (c) an antigen-binding fragment of an anti-KIM-1 antibody.

26. The method of claim 25, wherein the antibodies are of the IgG class.

27. The method of claim 26, wherein the antibodies are of IgG1 subclass.

28. The method of claim 27, wherein the effective amount of the polypeptide is administered to the mammal between 30 minutes and 30 days before the immune system of the mammal first recognizes the one or more antigens.

29. The method of claim 28, wherein the one or more antigens are alloantigens.

30. The method of claim 28, wherein the one or more antigesns are autoantigens.

31. The method of claim 28, wherein the immune system of the mammal first recognizes the one or more antigens as part of an epitope spreading process in the course of an autoimmune disease.

32. A method of inhibiting epitope spreading in an autoimmune disease, comprising administering administering an effective amount of a KIM-1 antagonist selected from the group consisting of: (a) a polypeptide comprising a KIM-1 Ig domain, and lacking a

transmembrane domain and a KIM-1 cytoplasmic domain; (b) an anti-KIM-1 antibody; and (c) an antigen-binding fragment of an anti-KIM-1 antibody.

33. A method of treating a Th2 cell-mediated disease, comprising administering an effective amount of a KIM-1 antagonist selected from the group consisting of: (a) a polypeptide comprising a KIM-1 Ig domain, and lacking a transmembrane domain and a KIM-1 cytoplasmic domain; (b) an anti-KIM-1 antibody; and (c) an antigen-binding fragment of an anti-KIM-1 antibody.

34. The method of claim 33, wherein the Th2 cell-mediated disease is selected from the group consisting of myasthenia gravis, autoimmune hemolytic anemia, Chagas disease, Graves disease, idiopathic thrombocytopenia purpura (ITP), Wegener's granulomatosis, polyarteritis nodosa, rapidly progressive crescentic glomerulonephritis, graft-versus-host disease (GVHD), and systemic lupus nephritis (SLE).

35. A method of inhibiting GVHD, comprising administering an effective amount of a KIM-1 antagonist selected from the group consisting of: (a) a polypeptide comprising a KIM-1 Ig domain, and lacking a transmembrane domain and a KIM-1 cytoplasmic domain; (b) an anti-KIM-1 antibody; and (c) an antigen-binding fragment of an anti-KIM-1 antibody.

36. A polypeptide comprising a KIM-1 Ig domain and an Fc moiety, and lacking a transmembrane domain and a KIM-1 cytoplasmic domain.

37. A method of inhibiting secretion of IFN-γ by lymphocytes in a mammal, comprising administering to the mammal an effective amount of a KIM-1 antagonist.

38. A method of treating an inflammatory disease or disorder in a mammal, comprising administering to the mammal an effective amount of a KIM-1 antagonist.

39. The method of claim 38, wherein the inflammatory disease or disorder is inflammatory bowel disease.

40. The method of claim 38, wherein the inflammatory disease or disorder is acute or chronic inflammation.

41. The method of claim 4, wherein the T helper cell is a Th1 cell.